Gastroesophageal Reflux Disease

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Several mechanisms work together to form a barrier against reflux. There are thickened muscle fibers in the distal esophagus that form the lower esophageal sphincter (LES). The diaphragmatic crura encircle the distal esophagus, providing further support for the LES. This creates a high-pressure zone that maintains a resting tone 10–30 mmHg above gastric pressure. The lower esophageal sphincter pressure (LESP) increases in response to situations where gastric pressure increases and relaxes in association with specific reflexes, such as swallowing, belching, and vomiting. The LESP is further supported by the transmission of abdominal pressure to the intra-abdominal portion of the very distal esophagus. The angle of His, an acute angle between the gastric cardia and the esophagus, creates a valve that contributes to the barrier against reflux. Lastly, esophageal motility clears refluxate from the lower esophagus with secondary peristaltic waves.

Gastroesophageal Reflux

Gastroesophageal reflux is the *effortless* passage of gastric contents up into the esophagus. It occurs when the mechanisms of esophagogastric competence malfunction or are overcome. Normal individuals experience GER without significant consequences. Gastroesophageal reflux disease (GERD) is defined as reflux that results in significant symptoms or harm to the patient. Development of GERD results from an imbalance between factors promoting reflux and the ability of the esophagus to clear and resist gastric acid exposure. The main factor leading to GERD is thought to be dysfunction of the LES complex.

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The most common mechanism of a reflux event is inappropriate transient relaxation of the LES, which is a sudden and brief decrease in pressure to near zero that is not triggered by swallowing. Transient relaxations occur in asymptomatic individuals, but they are more frequent in patients with GERD and account for the majority of reflux episodes. Less frequently, reflux episodes are associated with a reduced basal LESP.

Other factors predisposing to GERD include anatomic abnormalities such as a congenitally short esophagus, congenital diaphragmatic hernia, hiatal hernia, and esophageal atresia. Insertion of a gastrostomy tube may promote reflux by altering the angle of His, making it less acute or obtuse. In addition, esophageal dysmotility, delayed gastric emptying, elevated intra-abdominal pressure (peritoneal dialysis, gastroschisis/omphalocele, ascites, obesity), and central nervous system impairment are associated with an increased risk for symptomatic GERD.

Diagnosis

The most common symptom of GERD in children is regurgitation or vomiting of feeds. Other signs include fussiness with feedings, feeding refusal, and arching of the back during feeds (as in Sandifer syndrome). Consistent pain with feedings and feeding refusal may result in inadequate caloric intake and failure to thrive. Respiratory symptoms include reflex apnea, acute life-threatening events, and recurrent episodes of aspiration causing hoarseness, laryngitis, pneumonia, and chronic lung disease. Esophagitis may cause acute or chronic bleeding and pain and can lead to stricture formation and dysphagia.

While many children are treated medically for GERD based on history alone, without significant work-up, children who are being considered for surgery require thorough investigation to ensure that the symptoms are actually due to GER. Many of the symptoms of GERD can be due to other

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causes, such as central apnea, pharyngeal dysphagia, tracheoesophageal fistula, esophageal web, *Helicobacter pylori* infection, food allergy or intolerance, an intrinsically sensitive emetic reflex, gastric outlet obstruction (pyloric stenosis), or intestinal dysmotility.

We begin with a complete history and physical examination to determine the character, frequency, and severity of the patient's symptoms, as well as to rule out other potential etiologies. Accompanying symptoms such as pallor, salivation, sweating, or retching might indicate activation of the emetic reflex rather than passive GER. Several validated questionnaires exist for the detection and surveillance of GERD, which is often sufficient to diagnose uncomplicated GERD and initiate medical management.

When considering surgical management for GERD, our initial test of choice is a contrast upper gastrointestinal (UGI) fluoroscopic study. The sensitivity of UGI for detecting reflux is only about 30 % as compared to pH monitoring, partly due to the short duration of the examination. Specificity is also low, as the presence of reflux on UGI does not necessarily indicate pathologic GERD. Therefore, the decision to proceed with surgical treatment should not be based solely on GER found on UGI. However, this test does provide valuable information about the anatomy and can detect other abnormalities that may explain the symptoms or alter the surgical plan, such as malrotation, hiatal hernia, esophageal or duodenal web, achalasia, or pyloric stenosis. In the absence of these findings and a clinical picture of severe GERD refractory to medical treatment, UGI is often the only diagnostic test needed before proceeding to surgery in children.

If the diagnosis of GERD is uncertain, further diagnostic tests are warranted. Esophageal pH monitoring, the standard test for diagnosing GERD in adults, is used to quantify the amount of acid exposure to the lower esophagus. A drop in pH below 4.0 is considered an acid reflux episode. The number of episodes and their lengths are recorded to create a score reflecting reflux severity. The DeMeester score for adults and older children and the Boix-Ochoa revised score for infants and toddlers provide a quantitative assessment of acid exposure. Increasingly, pH monitoring is performed in conjunction with multichannel intraluminal impedance (MII), which uses multiple electrodes along the length of the esophagus and in the stomach to detect volume, velocity, and extent of fluid and solid boluses. This is particularly useful in detecting reflux of non-acidic material, which is more common in infants or patients on acid-suppressive therapy. When used in combination, pH monitoring and MII are useful to correlate documented reflux events with patient symptomatology.

Additional diagnostic tests that can be helpful include esophagogastroduodenoscopy (EGD) with biopsy, esophageal manometry, and technetium-99 radionuclide scanning to measure the rate of gastric emptying. In the setting of hematemesis, dysphagia, or a normal pH/MII study but continued suspicion of GERD, EGD can reveal esophagitis or Barrett's esophagus, confirming the diagnosis. If by EGD one finds evidence of gastritis, infection with *Helicobacter pylori*, or food intolerance such as gluten enteropathy, those conditions should be treated first and the patient reassessed before considering surgical intervention for reflux. Esophageal manometry, while considered essential in adults, is not commonly performed in the pediatric population. However, if the primary symptom is dysphagia, manometry can detect esophageal motility disorders such as achalasia. GERD can be associated with delayed gastric emptying, especially in neurologically impaired children. However, gastric emptying studies are not obtained routinely prior to surgical intervention, as gastric emptying rates have been shown to improve after fundoplication. Assessment of gastric emptying should be considered if symptoms persist after antireflux surgery or prior to undertaking a redo fundoplication for recurrent symptoms.

Medical Therapy

The majority of children with GERD respond to medical therapy, which may be attempted empirically, sparing patients from invasive diagnostic testing. The majority of infants respond to medical treatment and experience resolution of GERD symptoms by 12–18 months of age. Feeding behavioral modifications include smaller volume feeds in an upright position. In children receiving tube feedings, continuous drip rather than bolus feedings might help reduce the likelihood of reflux. Since milk-protein allergy may manifest with GERD-like symptoms, elimination of milk protein may be a helpful initial step. Additionally, food thickeners reduce the ease with which liquids reflux. In older children, dietary changes may include a low-fat diet and avoidance of caffeine and carbonated drinks.

If these maneuvers are unsuccessful, pharmacologic therapy may be attempted. The acidity of refluxed material is reduced using antacids, histamine receptor antagonists, or proton pump inhibitors (PPIs). These may dramatically decrease acid exposure, but will not address the effects of nonacid reflux. Additionally, though the efficacy of PPIs is well established in adults and older children, their use is somewhat more controversial in infants. Promotility agents, such as metoclopramide or erythromycin, are thought to ameliorate GERD, though their success has been mixed, and they are not without significant side effects, which limit their long-term utility.

Surgical Therapy

Surgical treatment of GERD is typically indicated if an adequate trial of medical treatment fails to control symptoms. Operative management without a trial of medical management may be acceptable in the presence of complications such as severe esophageal ulceration, Barrett's esophagus or stricture, chronic pulmonary disease or recurrent aspiration pneumonia, apparent life threatening event (ALTE) spells, or persistent failure to thrive. Neurologically typical children often improve with increasing age, and if symptoms can be controlled medically, we recommend a nonoperative approach. Surgical treatment is more frequently required in neurologically impaired children. Our preferred surgical management of GERD is the laparoscopic Nissen fundoplication, with or without simultaneous gastrostomy.

Most children can be admitted from home on the day of surgery. Rarely, children with chronic lung disease such as cystic fibrosis or spinal muscular atrophy require preoperative hospitalization for pulmonary toilet and intravenous antibiotics or TPN. Perioperative blood transfusions are typically not required. Because the surgeon's initial laparoscopic view of the stomach is from the umbilicus, up, and over the transverse colon, neurologically impaired children with chronic constipation and a chronically dilated colon may present an added challenge. Several enemas, administered in the evening before surgery, can help decompress the colon. A single dose of a first-generation cephalosporin is administered intravenously just prior to incision.

Laparoscopic Nissen fundoplication is a technically challenging advanced minimally invasive procedure. In general, the technical difficulty increases as the size of the patient decreases. Selecting laparoscopic instruments appropriate to the size of the individual patient is crucial. For infants and small children, we prefer 3-mm instruments that are available in varying shaft lengths of 10, 14, and 20 cm. These are inserted through stab incisions, without the use of a port. We use a 45° 4- or 5-mm laparoscope inserted through a 5-mm camera port located at the umbilicus. In older children, 5-mm instruments may be necessary as well as additional cannulas.

After intubation, the anesthesiologist inserts an orogastric tube, and the child is moved down toward the foot of the table. Infants and small children can be placed in a frog-leg position at the very end of the table, while larger children are positioned in lithotomy with stirrups. This allows the surgeon to stand at the foot of the bed or between the patient's legs. The scrub nurse stands to the surgeon's left and the assistant is to the right. One or two monitors are positioned at the head of the table.

After preparing and draping the abdomen, a 5-mm vertical incision is made through the center of the umbilicus and the camera port is inserted with open technique using a blunt-tipped cannula with trocar. The sheath can be sutured to the umbilical skin to prevent it from inadvertently sliding out. If a gastrostomy tube is to be placed, that site is marked prior to insufflation, as insufflation tends to distort the abdominal wall and its landmarks. That site is typically in the left upper quadrant, two fingerbreadths inferior to the costal margin and to the left of midline. After insufflating and visualizing the abdomen, a stab incision is made at that location for the surgeon's working right hand instrument, which is also where needles are introduced for intracorporeal suturing. The left-hand instrument is inserted through a stab incision in the right upper quadrant, just lateral to the falciform ligament near the inferior edge of the liver. A liver retractor is inserted through the right mid-abdomen, placed under the left lateral segment of the liver to expose the hiatus, and then secured to a post attached to the bed near the patient's right shoulder. The assistant's instrument is placed in the left lateral abdomen.

The operation begins with division of the short gastric vessels. The surgeon retracts the greater curvature of the stomach to the patient's right side, while the assistant grasps the vessels and provides countertraction to the left. Starting at the level of the inferior pole of the spleen, the vessels can be ligated and divided with monopolar electrocautery connected to a Maryland dissecting instrument in infants and young children. In older children, an ultrasonic scalpel or advanced bipolar device is used. As the superior pole of the spleen is reached, the assistant pushes the spleen to the left while the surgeon pulls the greater curvature of the stomach caudally to expose the most superior vessels. Once the spleen is separated, the left side of the hiatus is visualized. At this point, the dissection of a retro-esophageal window can be started from the left side, though often the left gastric artery is not clearly visualized. Minimal dissection is performed at the hiatus, leaving the phreno-esophageal membrane largely intact, which is important to reduce the risk of postoperative herniation of the wrap into the mediastinum and subsequent recurrent GERD.

The stomach is then retracted to the patient's left and attention turned to the right side of the stomach and hiatus. The thin gastrohepatic ligament can be entered with blunt spreading and then divided with electrocautery up to the level of the diaphragm to the right of the hiatus. The right side of the esophagus is identified as well as the left gastric artery. The retro-esophageal window is then completed, using two instruments to bluntly dissect the space cephalad to the left gastric artery, while the assistant grasps the phreno-esophageal fat pad. Again, unless there is a hiatal hernia, it is not necessary to dissect the native attachments between the esophagus and the hiatus to achieve adequate intra-abdominal length of esophagus in most children.

After completing the window, the right and left crura at the posterior hiatus often appear slightly separated behind

the esophagus. The hiatus is tightened by closing this defect with a single 2-0 silk suture on a ski needle. After tying the knot, the same needle is passed through the wall of the posterior esophagus at the seven o'clock position, with care to avoid the posterior vagus nerve.

After the crural repair, the fundus of the stomach is visualized through the retro-esophageal window, aided by the assistant pushing the fundus posteriorly and cephalad on the left side of the esophagus. With the assistant pulling the phreno-esophageal fat pad anteriorly and caudally, the fundus can be grasped through the window and pulled posterior to the esophagus, bringing it over to the right side to create the wrap. After ensuring appropriate positioning of the wrap, without twisting or tension, it is pushed back through the window to the left side while maintaining control of it with the left hand grasper. This allows visualization of the hiatus while a bougie is introduced into the esophagus by the anesthesiologist and advanced into the stomach. The bougie may catch on the posterior hiatus, which can be remedied by caudally retracting the phreno-esophageal fat pad to straighten the esophagus and placing a grasper up against the posterior hiatus. The appropriate bougie size is based on the child's weight (Table 43.1). The fundus is then pulled back through the window over to the right side. The wrap is completed with three sutures of 2-0 silk on a ski needle that are then tied intracorporeally. The superior most suture incorporates a small amount of the anterior esophagus and diaphragm at the 11 o'clock position, avoiding the anterior vagus nerve. The wrap is typically about 2 cm in length and should sit slightly to the right, with the sutures at the 11 o'clock position. The bougie is removed, local anesthetic instilled in the incisions, and the umbilical fascia and skin closed. The stab incisions can be closed with steri-strips alone.

If a gastrostomy button is to be inserted, it is placed though the left epigastric stab incision. With a single grasper through that incision, the greater curve of the stomach, across from the incisura, is grasped and pulled up to the anterior abdominal wall. Two transabdominal sutures of 0 or 2-0 polypropylene or polydioxanone suture (PDS) on a large curved needle are used to fix the stomach against the abdominal wall. These sutures are passed down through the abdominal wall, through the stomach medial and lateral to the grasper, then back up through the abdominal wall. A Seldinger technique is then performed using a vascular dilator set, which typically contains a needle, wire, and several dilators. The needle and wire are inserted into the stomach lumen and then the tract dilated up to 16 Fr. The wire and dilators can be gently swirled in a circular fashion to confirm intraluminal placement. To help select an appropriate size button, the tract length can be measured with a balloon-measuring device or estimated with the length of a grasper from the skin to the peritoneum. The smallest dilator can then be inserted through the button lumen and slid over the guide wire into the stomach and the balloon inflated under visualization. The site should be inspected to ensure the balloon did not inflate between the stomach and the abdominal wall. Placement within the lumen can be confirmed by insufflating air through the button into the stomach while visualizing distension of the stomach followed by decompression out through the button. The transabdominal sutures are then tied over the wings of the button to secure it in place. These sutures remain in place for 5 days postoperatively.

Table 43.1 Recommended bougie size for esophageal calibration in patients weighing less than 15 kg

Weight (kg)	Bougie size (Fr)
2.5–4.0	20–24
4.0–5.5	24–28
5.5–7.0	28–32
7.0–8.5	32–34
8.5–10.0	34–36
10.0–15.0	36–40

Source: From Ostlie DJ, Miller KA, Holcomb GW III. Effective Nissen fundoplication length and bougie diameter size in young children undergoing laparoscopic Nissen fundoplication. *J Pediatr Surg* 2002;37:1664–6, with permission from Elsevier

Postoperative Care

Feedings are typically started several hours postoperatively, whether by mouth or by gastrostomy. We advance tube feedings over the first night and first postoperative day, attempting to reach the goal rate by 24 h following the operation. Most children are able to be discharged home on the first or second postoperative day. Children who can eat orally are instructed to follow a liquid diet for at least 2 weeks to avoid food impaction above the Nissen. Edema of the Nissen wrap typically subsides over a period of 3–4 weeks. Occasionally, children with preexisting esophagitis require continuation of acid-suppressive medications for 6–8 weeks postoperatively to allow the esophagus to heal.

Neurologically impaired children and those with other complex medical illnesses often experience dysmotility of multiple sections of the GI tract and therefore are prone to postoperative digestive disturbances, including gas bloat syndrome, retching, high gastric residuals, and constipation. Retching and gas bloat symptoms can be reduced by slowing the rate of bolus feed administration and frequent venting of the gastrostomy tube between feeds. Some patients require a period of continuous drip feedings via the G-tube to minimize gastric distension. These symptoms typically improve with time.

Dysphagia can be a problem in the immediate postoperative period for children taking solid food orally. This is usually the result of edema of the wrap or possibly a wrap that is

too tight. If symptoms persist for more than 6 weeks, dilation will usually provide significant improvement, though this risks disruption of the wrap.

Results

The short- and long-term outcomes of laparoscopic Nissen fundoplication are excellent. Compared to open fundoplication, it is associated with shortened hospital stay, less pain, shorter time to goal feedings, and fewer postoperative pulmonary complications. While the rate of failure requiring reoperation historically has been reported at approximately 6–12 %, recent studies suggest that minimal dissection of the phreno-esophageal membrane lowers the reoperation rate to around 3 %. The risk of failure is increased if patients are younger and have retching or hiatal hernia and if the esophageal hiatus is extensively dissected at the first fundoplication.

Summary

Gastroesophageal reflux disease is primarily due to dysfunction of the LES-crus complex. Patients resistant to maximal medical therapy or with severe, life-threatening complications are candidates for antireflux surgery. Laparoscopic Nissen fundoplication is a safe, durable surgical option and has become the operation of choice for pediatric gastroesophageal reflux disease in most major children's hospitals.

Editor's Comment

GERD is one of the most frequent indications for referral to a pediatric surgeon. GE reflux is quite common in all humans, but there are certain children for whom reflux is severe and intractable or associated with complications such as pain, failure to thrive, aspiration, or reactive airways disease. It is important to distinguish reflux, which is effortless, from emesis, which is forceful. Fundoplications in patients with forceful vomiting always fail. The decision to operate should be based on clinical grounds. Ideally, there should be a consensus among the primary care physician, gastroenterologist, surgeon, and parents. It is unfortunate that in some centers there is a culture of distrust between gastroenterologists and surgeons, to the clear detriment of those patients who might benefit from an operation.

Objective testing is useful in some clinically borderline cases, but available tests are insensitive and nonspecific, and therefore cannot be used as the sole factor in making the decision. The only preoperative test considered mandatory by most pediatric surgeons is an UGI contrast study, which is useful not to confirm or exclude GERD but to rule out achalasia, esophageal stricture, and gastric anomalies and malrotation.

Neurologically impaired children often need enteral access for nutrition or medications but are also often unable to protect their airway, traditionally considered an indication for fundoplication, at the time of gastrostomy. Some families choose to forego fundoplication, especially if the child has been tolerating nasogastric feedings. This might be reasonable considering that these children also have the highest incidence of postoperative complications, retching, feeding intolerance, hiatal hernia, wrap failure, and recurrent reflux.

There are several time-tested principles that are critical to performing a successful fundoplication in children: (1) Perform a 360° wrap whenever feasible. Partial wraps are not as effective or as durable, though they might be preferable when esophageal motility is poor. (2) Close the hiatus by approximating the crura posterior to the esophagus. Anterior repair of the hiatus is ineffective as the stitches are doomed to cut through. The use of pledgets or mesh is associated with a risk of esophageal erosion and perforation and so should only be used if there is truly no alternative. (3) Make the wrap as loose as possible and be careful to avoid twisting the stomach around the lower esophagus, which causes severe dysphagia. Always use a bougie to prevent overtightening of the hiatus or the wrap. There are published weight-based guidelines for the appropriate bougie to use, but one should use the largest bougie that the esophagus will accommodate comfortably. (4) Mobilize at least 3 cm of esophagus into the abdomen to make a wrap 2–2.5 cm in length. Avoid dissection of the phreno-esophageal ligament as this results in trans-hiatal migration of the wrap in up to a third of patients. Use at least three braided permanent stitches and include a bite of the esophagus with each stitch. Identify and protect both vagus nerves throughout the procedure to minimize gastroparesis. (5) Avoid unnecessary stitches—collar stitches between the esophagus and hiatus, "rip-stop" stitches (fundus to fundus below the lowest wrap stitch), or stitches between the fundus and diaphragm—which are only useful in unusual situations. (6) Always divide at least some of the upper short gastric vessels. This allows more of the fundus to be wrapped and the creation of a tension-free wrap. (7) Use a minimally invasive approach whenever possible, except maybe in small infants and some redo operations, as for many reasons it is clearly superior to the open procedure. The standard position is for the surgeon to stand at the foot of the table between the legs in stirrups, but experienced laparoscopists can easily perform the procedure standing next to a patient who is supine.

Intraoperative complications are rare but can be serious. One should be wary of an accessory or replaced left hepatic artery. If a particularly large vessel is "in the way," it makes sense to test-clamp it to be sure the liver does not demarcate. Passing a bougie can perforate the esophagus and should be considered the most dangerous part of the operation. The surgeon and anesthesiologist must agree that the bougie should be advanced slowly and only when both parties are

aware. Most perforations are low and small and best repaired primarily and covered with the wrap. Perforations of the thoracic esophagus are best managed by aborting the fundoplasty, establishing adequate drainage, restricting PO intake, and obtaining an esophagram at 5–7 days.

Redo fundoplication can be extremely tedious, mostly due to dense adhesions. This is considered by some surgeons to be an added advantage to the laparoscopic approach: revising the wrap is somewhat easier and can often be done again laparoscopically. The vagus nerves are at high risk for injury during revision fundoplasty, but performing an empiric pyloroplasty is no longer recommended due to the risk of dumping syndrome. Finally, when revising a fundoplication, it is important to take it down completely first, rather than simply reinforcing the part that has loosened. This allows proper closure of the hiatus, identification of the reason for failure, and creation of a tension-free and hopefully more durable wrap.

Postoperative dysphagia occurs in approximately 10 % of patients after fundoplication, but only about 10 % of these persist for more than 6 weeks. Those that persist should be considered for dilatation of the fundoplication, best done using a balloon dilator under fluoroscopic guidance. Refractory dysphagia is rare but will usually require revision, conversion to a partial wrap, or undoing the wrap.

Suggested Reading

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